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UWill Discover Undergraduate Conference

UWill Discover 2018

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Mar 22nd, 9:20 AM - 10:40 AM

### The Role of Spy1 in Glioblastoma Multiforme Initiation and Progression

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El-Abed, Youshaa; Qemo, Ingrid; Porter, Lisa; and Lubanska, Dorota, "The Role of Spy1 in Glioblastoma Multiforme Initiation and Progression" (2018). *UWill Discover Undergraduate Conference*. 1.  
<https://scholar.uwindsor.ca/uwilldiscover/2018/all2018/1>

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Glioblastoma Multiforme (GBM) represents the most common and aggressive form of brain tumour. The success of therapeutic approach has been hindered by the extreme heterogeneity observed not only between individual patients but also within a single tumour. It has been shown recently that certain cell populations within the tumour possess stem cell properties contributing to the heterogeneity, aggressive character and therapy resistance of GBM, a phenomenon known as cancer stem cell hypothesis. Deregulation of the cell cycle control network plays a critical role in maintaining proliferation and stem like characteristics of cancer cells in GBM. In addition, mutations and/or deletions of tumour suppressors p53 and pTEN are some of the most common features of stem like cell population of brain tumours. Speedy (Spy1) is a cyclin-like protein that has been shown to enhance cellular proliferation and stem cell self-renewal in several systems, including brain. Moreover, Spy1 has been shown to be upregulated in GBM and elevated levels of Spy1 are indicative of poor prognosis of patient outcome in GBM. A mouse model, termed NTA-Spy1, was used in order to overexpress Spy1 in the specific stem cell population in the brain. To date, NTA-Spy1 mice have shown no spontaneous tumour formation. When these cells or their controls are combined with knockdown of either p53 (shp53) or drug inhibition of pTEN (pTENi) alone, or in combination, NTA-Spy1 cells increase the rate of tumoursphere formation in soft agar cell cultures. It also has been shown that NTA-Spy1 cells in combination with shp53 or pTEN inhibitor increases gene expression of GBM cancer stem cell markers. This work will help explain the role of Spy1 in susceptibility to brain tumour initiation and progression and the importance of its overexpression in face of other aberrant. This research may provide insight into novel targeted therapies that could be designed against GBM.